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Whole body atrophy.

Atrophy in general is related to changes in nutrition and metabolic activity of cells and tissues. A widespread or generalized **atrophy** o body tissues occurs under conditions of **starvation**, whether becaus food is unavailable or because it cannot be taken and absorbed due to the presence of disease. The unavailability of certain essential protein components and vitamins disturbs the metabolic processes and leads to **atrophy** of cells and tissues. Under conditions of prote starvation, the body protein is broken down into constituent amino acids, which serve to provide energy and help maintain the structu and cells of the most essential organs. The brain, heart, adrenals, thyroid, pituitary, gonads, and kidneys show less **atrophy**, relativel than the body as a whole; whereas the fatty stores of the body, liv spleen, and lymphoid tissues diminish relatively more than the bod as a whole. The brain, heart, and kidneys, organs with abundant blood supply, appear to be the least subject to the wasting effects starvation.

Associated with the widespread **atrophy** due to lack of protein is th **atrophy** of certain tissues that is due to deficiencies of specific vitamins. Atrophic changes of the skin increase because of the lack vitamin A, and **atrophy** of muscle increases because of the unavailability of vitamin E.

After a growth period of human metabolism, there sets in a gradua decline: slow structural changes other than those due to preventab diseases or accidents occur. **Aging** eventually is characterized by marked **atrophy** of many tissues and organs, with both a decline in the number of cells and an alteration in their constitution. This is reflected eventually in the changed, diminished, or lost function characteristic of old age and eventuates in death. The changes in

senescence are affected by both inherited constitution and environmental influences, including disease and accident.

Atrophic changes of aging affect almost all tissues and organs, but some changes are more obvious and important. Arteriosclerosis—th thickening and hardening of arterial walls—decreases the vascular supply and usually accentuates aging processes.

Atrophy in old age is especially noticeable in the **skin**, characteristically flat, glossy or satiny, and wrinkled. The **atrophy** due to aging changes in the fibres of the true skin, or dermis, and i the cells and sweat glands of the outer skin. Wasting of muscle accompanied by some loss of **muscular** strength and agility is common in the aged. In a somewhat irregular pattern, there is shrinkage of many individual muscle fibres as well as a decrease in their number. Other changes have been observed within the muscle cells.

Increase of the pigment lipofuscin is also characteristic in the musc fibres of the **heart** in the aged in a condition known as brown **atrop** of the heart. Wasting of the heart muscle in old age may be accompanied by increase of fibrous and fatty tissue in the walls of the right side of the heart and by increased replacement of elastic tissue with fibrous tissue in the lining and walls of coronary arterie within the heart muscle. Abnormal deposits of the protein substanc amyloid also occur with greater frequency in the atrophic heart muscle in old age.

Atrophy of the **liver** in the aged is also accompanied by increased lipochrome pigment in the atrophied cells.

The bones become progressively lighter and more porous with aging a process known as **osteoporosis**. The reduction of bone tissue is most marked in cancellous bone—the open-textured tissue in the e of the long bones—and in the inner parts of the cortex of these bon In addition to changes in and loss of osteocytes, or bone cells, ther is decreasing mineralization, or calcium deposit, with enhanced fragility of the bones.

Atrophy of the **brain** in old age is shown by narrowing of the ridges or gyri, on the surface of the brain and by increased fluid in the sp beneath the arachnoid membrane, the middle layer of the brain covering. There is shrinkage of individual **nerve cells**, with an increase in their lipochrome pigment content, as well as a decrease in their number. Sometimes the nerve fibrils have degenerated, an deposits called senile plaques may be found between the nerve cel particularly in the frontal cortex and hippocampus (a ridge in the w of an extension, or horn, of the lateral ventricle, or cavity, of the

brain). Similar atrophic changes are seen in the brain in Alzheimer' disease, a condition of unknown cause most likely to occur in older patients. The mental deterioration (senile dementia) of the aged is the clinical manifestation of these changes. Senile **atrophy** may be increased and complicated by the presence of arteriosclerosis.

Simmonds' disease is a chronic deficiency of function of the pituita gland that leads to **atrophy** of many of the viscera including the heart, liver, spleen, kidneys, thyroid, adrenals, and gonads.

A destructive or atrophic lesion affecting the **pituitary glands** with loss of hormones leads to **atrophy** of the thyroid, adrenal glands, a gonads and in turn brings atrophic changes to their target organs an the viscera. The decrease in size of the endocrine glands may be extreme.

Atrophy of muscle or of muscle and bone.

Local **atrophy** of muscle, bone, or other tissues results from disuse diminished activity or function. Although the exact mechanisms are not completely understood, decreased blood supply and diminished nutrition occur in inactive tissues. Disuse of muscle resulting from loss of motor nerve supply to the muscle (*e.g.*, as a result of poliomyelitis) leads to extreme inactivity and corresponding **atroph** Muscles become limp and paralyzed if there is destruction of the nerve cells in the **spinal** cord that normally activate them. The shrinkage of the paralyzed muscle fibres becomes evident within a few weeks. After some months, fragmentation and disappearance o the muscle fibres occurs with some replacement by fat cells and a loose network of connective tissue. Some contracture may result.

The skeletal tissues forced to inactivity by **paralysis** (*e.g.*, of a limb as a result of poliomyelitis) also undergo disuse **atrophy**. If there is tendency for bone to become lighter and more porous in some particular area, a condition known as local osteoporosis, this can b recognized by X-rays within a few weeks. The cortex of the long bones becomes considerably thinned or atrophic, with decreased mineral content. Disuse as a result of painfully diseased joints, as i **rheumatoid arthritis**, results in a similar but lesser degree of **atrophy** of muscles concerned with movement of the involved joint and local **atrophy** may also occur in the bone in the neighbourhood the joint. A local osteoporosis of bone known as Sudeck's **atrophy** sometimes develops rapidly in the area of an injury to bone.

Severe or prolonged deficits of blood sugar deprive the nervous system of needed sources of energy and as a rare event result in degeneration of cells of the brain and peripheral nerves. The disuse **atrophy** of muscle or bone that may result is fundamentally similar

the other disuse atrophies of these tissues.

Persistent pressure will cause atrophy of a compressed cell, organ or tissue, presumably because of interference with the nutrition an metabolic activity of the affected part. Cells in a local area (*e.g.*, the liver) atrophy from the pressure of materials such as amyloid deposited around them. The pressure of an expanding benign tumo causes atrophy of adjacent normal structures. The pressure of a localized dilatation of an artery (aneurysm) will cause atrophy of tissues, even bone, on which it impinges.

Bulging of an intervertebral disk or growth of a tumour sometimes brings pressure on nerves near their point of exit from the spinal cord; if the pressure is prolonged, the muscles normally controlled these nerves may atrophy. Most often the calf muscles are affecte Pressure as a result of involvement of the vertebrae at the level of the neck, or from compression of the network of nerves called the brachial plexus by the *scalenus anticus* muscle, produces similar effects in the upper chest and arms.

Simple disuse of muscle or bone, as, for example, from the immobilization produced when a limb is put in a cast or sling, resul in atrophy of these tissues. In the case of muscle, the degree of atrophy is generally less severe than that caused by injury to a ner although the nature of the change is similar.

Localized atrophies of leg and arm muscles may result from hereditary or familial diseases in which the nerves of the spinal cor that supply them are inactivated or destroyed. In Charcot-Marie-Tooth disease, the atrophy involves mainly the peroneal muscles, the outer side of the lower legs, and sometimes the muscles of the hand as well. It commonly begins in childhood or adolescence. Peroneal muscle atrophy is also seen in the hereditary spinal cord degenerative disease known as Friedreich's ataxia.

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